Vol. XXXIII., No. 8. AUGUST, 1916. Whole No. 392

THE AMERICAN JOURNAL OF OPHTHALMOLOGY

EDITED AND PUBLISHED MONTHLY BY

ADOLF ALT, M. D.

ASSISTANT EDITOR.

W. F. HARDY, M.D.

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316 METROPOLITAN BUILDING. ST. LOUIS, MO.

Entered at the Postoffice at St. Louis, Mo., as Second-Class Matter.

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OF
OPHTHALMOLOGY

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ORIGINAL ARTICLES.

HISTOLOGIC EFFECTS OF HEAT ON THE EYE.

By Wm. E. Shahan, M.D., and Harvey D. Lamb, M.D.,
ST. LOUIS, MO.

From the Laboratory of Ophthalmological Pathology, Washington University.

The following observations were made on rabbit eyes heated by direct application by means of a thermophor, as described in the article, "Effects of Heat on the Eye," published in the *Journal of the American Medical Association*, August 5, 1916.

Very briefly the thermophor consists of a metal tube containing a thermometer, and surrounded by a resistance coil to generate heat, while within the latter is placed a zinc-iron sensitive strip to permit the temperature to be kept at any constant point. Into one end of the metal tube are inserted applicators of various shapes, to be applied directly to the cornea.

The right eye of rabbit No. 2 was heated at 136 degrees F. for 10 minutes under general anæsthesia (ether), with an applicator 7 mm. in diameter applied to the upper middle portion of cornea. Immediately following the heating there was noticed a moderate clouding and loss of epithelium in the heated area. The epithelium was restored in 3 days, but the cloudiness persisted. At the end of one week there was a beginning keratoconus and an invasion of bloodvessels into cornea from above. The keratoconus increased until the 12th day, and then diminished, having about disappeared at end of three weeks. On the 15th day the iris opposite the heated spot showed a whitish area which remained. At the end of four weeks the keratoconus was gone, a vascular and somewhat pigmented scar occupied the center of the heated area and the upper third of the iris was gray. The eye was enucleated and fixed in formalin.

Microscopical findings.—Sections through this eye show the thickness of the cornea increased through its upper half, reaching a thickness through the upper fifth of the cornea of about 1½ times the normal depth. This increase in thickness is close to the newly formed connective tissue, just beneath the epithelium, containing several small capillaries, but few lymphocytes, leucocytes or plasma cells.

The posterior endothelial layer in the upper fifth of the cornea is rather thickly stippled on its surface with groups of small light brown pigment granules. This pigment, as we shall soon see, has undoubtedly come from the upper part of the iris. That it should be deposited here more thickly seems difficult to explain, unless this layer presenting a rather rough surface in its process of regeneration at this spot caught more of the floating pigment. This upper position of the posterior layer, it is true, would have the first chance at the pigment from the iris in our case since, according to the observation of Berg, there is a current upward in the posterior part of the anterior chamber, and one downward just behind the cornea.

The changes in the iris are much the most interesting. As noted above, the iris in its upper part was before death of a whitish appearance. We now see this to be due to a destruction of the cells in the iris stroma and a rearrangement of the pigment. (Where the iris has not been affected by the heat, a rather dense stroma is seen containing many stellate richly pigmented cells generally distributed, and in addition forming a thin layer just beneath the anterior endothelium and around the larger blood-vessels.)

In a dense, poorly staining fibrous structure are seen a large number of round or oval shaped cells, some black with contained pigment, others less densely filled, being dark or light brown. Clumps of these pigment cells are also seen here and there just in front of the post-epithelial layers, and here without doubt these cells are epithelial in origin, having simply migrated from the retinal pigment layer. However it must be that others of these cells, amœboid and phagocytic in character, are either plasma cells or enlarged polymorphonuclear leucocytes, which have taken up part of the pigment set free by the destruction of the stellate chromatophores. Some of this pigment, however, we see in a granular form lying more or less thickly between the large pigment cells. In many places some of these latter seem to be in the process of being taken up by phagocytic cells. The anterior endothelial layer seems almost everywhere intact.

The left eye of rabbit No. 2 was heated for 10 minutes at a temperature of 129 degrees F., using an applicator 7 mm. in diameter applied over the upper middle portion of the cornea. Immediately following there was an opal white clouding over the upper half of the cornea with loss of epithelium. The epithelium was restored in three days. A whitish atrophic area of the iris at the upper border of the pupil was noted after two weeks. At the end of three weeks the cornea was clear, except for slight pigmentation over the heated portion. A small gray cloud was formed in the anterior capsule of the lens, which had increased in density by the 26th day.

On the 54th day the upper part of the cornea was slightly cloudy and vascular; the upper part of the iris grey. The eye was removed and fixed in formalin.

Microscopical findings.—The cornea for about 1 mm. from the upper limbus shows the formation of a thin layer of scar tissue just beneath the epithelium containing many bloodvessels some deeper ones quite large. Infiltrating this tissue are many leucocytes and some large lymphocytes and plasma cells; the leucocytes being present in great numbers in the subconjunctival tissue at the adjacent limbus.

Descemet's membrane and the posterior endothelial layer show no changes. The pupillary edge of the iris above appears thickened and bulbous for about a third of the width of the iris where the same changes occurred as described for the right eye. The retinal pigment layer here however ends before reaching the pupillary edge, being partly replaced by small clumps of the round or oval pigment cells. The anterior endothelial layer is wanting over the margin of the iris where the heat effects were most marked.

An applicator with point 7 mm. in diameter was applied to the left eye of rabbit No. 3 for 10 minutes at a temperature of 132 degrees F. Slight cloudiness was caused which increased in density in the next few days. On the third day a purulent discharge was noted and on the 5th day a slight hypopyon; no staining with fluorescein. The hypopyon disappeared by the 7th day, although the iris appeared inflamed. On the 10th day the eye was quieter, the iris above buckled and grey looking, bullous keratitis over the spot of the former infection. On the 54th day the eye was quiet, a thin scar occupied the central part of the cornea, the upper part of the iris was grey and retracted upward. The eye was enucleated and fixed in formalin.

Microscopical findings.—Just above the center of the cornea is seen a stretch including about one-fourth the diameter of the cornea where its thickness diminishes progressively to little more than half its ordinary thickness, and then increases again. The thinnest middle third of this stains more deeply with eosin, and seems more contracted. A thin layer of scar tissue lies just beneath the anterior epithelium over this quarter of the cornea. The margins of this stretch show a moderate amount of leucocytic infiltration. At the lower margin nuclei of polymorphonuclear leucocytes are seen in process of degeneration. At the upper margin, nearest the limbus, are seen many leucocytes, some plasma cells, all well preserved, being near several deep-lying bloodvessels. The cornea above the superior margin is vascular and infiltrated with leucocytes for about half its thickness. The vessels in the neighboring subconjunctival tissue are quite hyperæmic. The anterior epithelium over this superior margin is much roughened and in places the layers are but loosely connected, as we should expect from the formation of vesicles noted. This irregularity and looseness of the epithelial layers continues up to the superior limbus, the thickness of the epithelium in this upper quarter, although very irregular, being less than elsewhere.

The iris in this eye in its entire width above the pupil is thickened and changed. This thickening has resulted in applying the root of the iris against the cornea, thus in this spot closing the filtration angle. The density of the fibrous tissue in the changed stroma of the iris can in this eye be made out quite clearly since it takes the eosin more deeply. In this tissue we see the same round or oval pigment-filled cells mingled with some plasma cells and stellate chromatophores, between which in many places is seen much granular pigment. The anterior endothelial layer over the inner third of the iris has disappeared for the most part.

The left cornea of rabbit No. 4 was heated for 10 minutes at a temperature of 124 degrees F. with a 7 mm. applicator. The epithelium was removed over an area 7 mm. in diameter with no clouding. On the next day there was clouding of the cornea and the epithelium was gone over an area 5 mm. in diameter. Eye enucleated.

Microscopical findings.—The cornea in its upper half is thickened slightly, and here are noticed on its anterior surface the presence of a few deep depressions, becoming most noticeable at about 3 mm. from the superior limbus where they produce a scal-

loped appearance. This is undoubtedly due to the shrinking in the alcohol after an immediate initial swelling caused by the heating.

The cornea over the quarter just above the center shows a loss of anterior epithelium. The epithelium adjoining this denuded area has a rather irregular and loose formation as if newly formed, most noticeable on the superior side where it extends to about 2 mm. from the limbus. Bowman's membrane in spots near the center of the denuded area is broken up and roughened. This portion of the cornea uncovered by epithelium in its anterior half is irregularly and moderately infiltrated with polymorphonuclear leucocytes, most of which are elongated and flattened as they lie squeezed between the corneal layers. Posteriorly the endothelial layer shows a destruction of its cells beneath the heated area extending above to within 2 mm. of the superior limbus. Although completely gone in some places, in others there remains of these cells some loose cytoplasm lying on the posterior cell membrane, anterior to which lies the intact Descemet's membrane. Several small groups of leucocytes are seen lying here and there on Descemet's membrane or on remnants of the endothelium. No further changes can be noted in the iris or elsewhere.

The right eye of rabbit No. 6 was heated at 119 degrees F. for 10 minutes with a 7 mm. applicator. The epithelium was removed in patches over an area 8 mm. in diameter. Eye enucleated.

Microscopical findings.—The cornea is slightly thicker in the heated area. In the center of the cornea a stretch of about 2 mm. is quite devoid of epithelium. The anterior surface of this uncovered portion shows many indentations, giving an irregular scalloped appearance. Bowman's membrane is entirely intact, as are Descemet's membrane and the posterior endothelial layer. No cellular infiltration (for which there has not been time) has occurred. At neither limbus can there be seen any reaction whatsoever. No changes can be noted in the other structures.

We might therefore conclude from our findings that the immediate effects of the heated applicator upon a rabbit's eye are first a swelling of the heated portion of the cornea due to a localized œdema, which with increase of heat is accompanied by destruction of the anterior epithelium, then a destruction of the posterior endothelial layer, further by a coagulation necrosis of Bowman's membrane and anterior layers of the substantia pro-

pria of the cornea, and lastly a swelling and coagulation necrosis of the iris. Later effects are: first, the appearance of the infiltration of the anterior layers of the cornea with polymorphonuclear leucocytes, then the formation of scar tissue and new bloodvessels to replace the destroyed substantia propria of the cornea. Lastly, the new connective tissue with its round pigment cells, where before was iris stroma.

CONTRIBUTION TO THE ÆTIOLOGY OF THE SPONTANEOUS ABSORPTION OF CATARACT.*

By Dr. L. Verrey,
LAUSANNE.

In the September, 1913, number of the *Archives d'Ophtalmologie* I have related a case of spontaneous total absorption of a cataract; V.=1/6, came on without operation after a period of blindness lasting 21 years. The second eye had been operated on 25 years previously and the beginning of the cataract had shown itself when the patient was barely 40 years old. The eyes were strongly myopic.

I remarked on this subject, that in my case as in many of the published ones up to now, the spontaneous absorption had taken place in pathologic eyes and after attacks of iritis and glaucoma. I concluded my paper by drawing attention to this fact, in which I saw a relation of cause and effect, not forgetting that the mechanism of this absorption or the phases through which it passes are as yet little or badly known.

I have lately had a chance to observe a patient in whom it was plain that I was in the presence of a phase of transition of a ripe cataract to its absorption. It seemed that the latter might become complete with spontaneous restitution of vision any day under the influence, for instance of a slight trauma.

The following is the history:

M. G. J., 56 years old, consulted me in October, 1915, asking me to operate on his left eye for cataract. The examination reveals numerous posterior synechiæ, the lens capsule is very dark and thick throughout the pupillary area. The fundus cannot be illuminated. Light projection is good.

*La Clinique Ophtalmologique, April, 1916.

Operation on October 25th, 1915, with the assistance of my son. Fearing a thick secondary cataract on account of the thickened anterior lens capsule I tried to remove this with capsule forceps, but could not get a hold of it without having first made an iridectomy. I then made an iridectomy and capsulotomy with the ordinary kystitome. Hardly was the capsule opened, when we saw a quantity of an almost clear liquid flow out, but no nucleus as in an ordinary Morgagnian cataract. At the same time a membrane presented between the lips of the corneal incision which I grasped with forceps and pulled out in toto without having to cut it off with the scissors. No vitreous body was lost. At the first inspection this membrane appeared to form the greatest part of the lens capsule, except a small portion of the posterior capsule. It had the shape of a sac in which we found a very small nucleus of the size of a very small flattened pin head. The fundamental difference from what we see in Morgagnian cataract lies in the fact that, contrary to what we find in this form of cataract in which the whole capsule is thickened, in our case there was a thickening only in the pupillary part of the anterior capsule.

The consequences of the operation were fairly normal. Yet, the cornea remained disturbed for a number of days, as if the eye in spite of its large anterior opening was in a state of tension. As usual I had made a large conjunctival flap and under this reapplied flap the gaping wound could be seen so that the eye had to remain bandaged for more than ten days, in spite of the bandage we discovered on the seventh day a hæmorrhage into the anterior chamber. When after a few days this was absorbed its remnants were seen hanging down from above in the region of the ciliary body and infiltrating the anterior layers of the vitreous body.

Under these conditions vision remained bad, and when the patient left the hospital he counted fingers only at two or three metres. When I saw the patient several weeks later V. was only 1/10 with +12 D. There seemed to be in the pupillary area and especially in its upper part some disturbance localized in the anterior layers of the vitreous body. The question was whether this would disappear spontaneously and whether a discission could be made after a few months The future would have to teach.

Concerning the operation itself we see how useful is a flap of conjunctiva cut in this membrane after the corneoscleral incision.

After having done this for about 20 years, followed Pansier's method for several years and having practiced the method with a conjunctival bridge, I left this latter very quickly on account of the difficulty in expelling the lens and especially in making the toilette of the anterior chamber. Then I commenced to practice a method similar to the one called of Truc, that is cutting a larger or smaller conjunctival flap according to the possibility in each case. I do not know whether this was before or after Truc's publications. At any rate the presence of a conjunctival flap, which is reapplied solidly to the wound already in twenty-four hours, impedes the infection of the corneoscleral wound.

Mr. G. tells me that he called on Professor Gayet, of Lyon, to have his right eye operated on for cataract. Before deciding on an operation Mr. G. had undertaken different treatments in order, as he was told, to absorb the cataract. The oculists or physicians he had seen during this time had at different occasions mentioned some adhesions of the iris and an increased intraocular tension. The treatment consisted of subconjunctival injections of mercury bichloride, mercurial inunctions, Gibert's syrup, instillations of different kinds. All these treatments were meant to act against attacks of iritis and glaucoma. It seems, therefore, that when Mr. G. called on Professor Gayet in 1891 the condition of his right eye was identical with the one I saw in the left eye in 1915. When Mr. G. had noticed his left eye becoming affected he treated it himself with different instillations. It does, however, not seem probable that these treatments pursued during the period of ripening of the cataract of either eye had any influence on the liquefaction which took place. In fact, having finished the operation, Professor Gayet said to Mr. G.: "I cannot show you your cataract; there was none. Give me the pleasure when your other eye gets affected to come back to me for operation. Yours is a very rare and curious case." From this we may conclude almost with certainty that in 1891, as in 1915, the cataract had become liquefied with total or almost total disappearance of the nucleus.

Regarding Morgagnian cataracts Panas (*Traité des maladies des yeux*, V. I, p. 541) made an observation which is of particular interest for us. He says: "In exceptional cases the fluid is clear and transparent like water, so that it looks as if the whole cataract had become absorbed; but by a careful examination under oblique light, we find in the lower part of the pupil, become black again, a small amber colored nucleus simulating a dislocated lens . . . ."

In distinction from traumatic cataracts due to perforating injuries Louis Dor says in his article on acquired cataracts (Vol. III, *Encyclopédie Française d'Ophtalmologie*): "Inflammatory cataracts, so called, when there is no inflammation from the outside which produces the increased volume and resistance of the capsule, but an inflammation of the subcapsular cells. We might for such cases re-establish the forgotten name of phakitis. This variety of capsulolenticular cataract is found especially in cases in which there has been an inflammation, that is after a trauma, after microbic affections and secondarily to a choroiditis and iritis."

Mr. G., to-day 56 years old, was hardly 30 years old when he was operated on by Gayet. The cataract was, therefore, not a senile one, but an inflammatory cataract, the treatment previously adopted to cure an inflammation of the affected eye prove it. Although developed many years later the cataract in the left eye has evidently the same ætiology. It appears then that in certain forms of inflammation of the internal membranes of the eye, of the iris, choroid, ciliary body, of an infectious character, from grippe or otherwise, with or without the tendency to an increase of the intraocular pressure, infectious elements can pass through the lens capsule which produce, what cannot be named better by any other name than the one recalled by Dor: a *phakitis*. This phakitis gives rise to the opacification of the lens fibres and by a chemical process which we do not yet know, in a certain number of cases only, to the liquefaction of the opacified lens fibres. This liquefaction is followed by a progressive absorption of the greatest portion of the albumins because finally nothing remains in the capsule besides a clear and transparent fluid. If at this time a fissure or tear occurs in the capsule, either due to an external traumatism, or spontaneously under the influence of the pressure of the fluid within the capsule, if even so slight that it may not have been noticed, the capsule is emptied of its contents; then the capsule may retract according to its elasticity as is the case after a discission, and all of the lens that remains in the pupillary area is a little thin membrane formed by the posterior capsule. The pupil has spontaneously been cleared up, vision is re-established to a certain degree, and thus we have seen the different phases by which nature has been able to restore the function of vision without any outside intervention.

In a paper on spontaneous absorption of cataract in the *Arch. d'Ophtalmologie*, September, 1913, I have tried to draw attention

to the frequency with which these cases with inflammatory phænomena, iritis, iridochoroiditis, glaucoma, are noted by the authors. The case which I related was of the same kind, that of a man, operated on twenty years previously for cataract and whose second eye had become affected only a little later, had at different times been suffering from slight iritis, glaucomatous attacks, and who finally in the space of six months had noticed a spontaneous clearing up of this eye and a return of vision of 1/6 of normal. Excepting a little opacity in the pupillary area, undoubtedly due to the presence of the posterior capsule, nothing could be seen, especially no remnant of a nucleus. I cited the words of Reuss:* "When reading all of the published cases one is struck by the fact that in more than half of these cases there were ocular complications; glaucoma in seven cases; in an eighth case a glaucomatous excavation was found after the absorption had taken place; in Reuss' first case an iritis, in Czermak's old posterior synechiæ; in Vossius' case the absorption coincided with an attack of iridocyclitis; in Berlin's case, iritis; in Hilbert's old foci of choroiditis; the same in Reuss' second case; with the patients of del Monte, Brettauer and Avitvalsky many opacities in the vitreous body. In fact in 34 cases of spontaneous absorption, complications were found in 15." I will add to what Reuss stated that if attention had been paid earlier to this point it is not impossible that in a goodly number of the other cases in which no inflammatory complications have been noted, they may have nevertheless existed. Bibliographic researches made in the literature since 1900 when Reuss' paper had appeared, did not discover any new cases in which the spontaneous absorption was accompanied by inflammatory phænomena. I cited those of Trousseau (*Annal. d'oculist.*, 1884) after glaucoma; Natanson (*Klin. Mtsbl. f. Aughlk.*, XXXIV and XXXIX) 33 cases of spontaneous absorption; Cazalis (*Ann. d'oculist.*, 1905) absorption of cataract after glaucoma; Augieras (*Clin. opht., Aug.*, 1904) two cases of spontaneous absorption, one of which was preceded by a serous iritis; Harms (*Klin. Mtsbl. f. Aughlk.*, Vol. XLIII, February) pathological anatomical examination of an eye with glaucoma in which the lens was half absorbed and dislocated.

The presence of residues of an iritis, foci of choroiditis, vitreous body opacities, lesions likely to give rise to an increase in intraocular pressure, especially when accompanied by opacity of

*Centrlbl. f. prakt. Augenhlk., February, 1900.

the lens fibres and swelling of this organ is, in my opinion, more than an accidental coincidence. We must see in this the relation of cause and effect. We should not be astonished, now that attention has been called to this point if in the large number of cases which will be published in the future, similar complications will be noted by the authors as having preceded the absorption. If this relation of cause and effect is confirmed and if the spontaneous absorption must be considered as a consequence of these inflammatory ocular phænomena, preceding or accompanying the formation of the cataract we may, perhaps, be able to give to this sequence of phænomena a scientific explanation which will amount to more than a simple theory. At present we must be satisfied to draw from the preceding facts the following simple conclusions:

The spontaneous absorption of a cataract, usually taking a long time, is in by far the greatest number of cases preceded by inflammatory phænomena, iritic or otherwise. The intraocular infection probably penetrates through the lens capsule, giving rise to a true "phakitis," followed by swelling of this organ frequently accompanied by an increase of the intraocular pressure, the liquefaction of the lens fibres, more or less complete absorption of the nucleus even, and finally in the fortunate cases rupture of the capsule, its retraction and the formation of a black pupil with restitution of vision to a varying degree which, however, never seems to reach more than 1/6 of normal.

Can we for the future draw any practical conclusions from these statements? and, can we ever obtain a spontaneous cure of cataract by means similar to those which nature employs in order to reach her object, that is a more or less complete restitution of vision by her own forces, by abolishing the visual function during a longer or shorter period?

I confine myself to asking this question. Time must answer it!

ON THE CAUSES OF PRESBYOPIA AND CATARACT.*

By Dr. F. Schanz.

With age a harder nucleus is formed in the center of the crystalline lens. Moerner† has shown that we must not see the difference between the nucleus and the cortex in the watery contents, but in that the nucleus contains more insoluble material than the cortex. He draws attention to the similarity with the skin. With age the cells of the skin contain more keratin; in the same manner the fibres in the interior of the crystalline lens contain more insoluable substances than the younger fibres of the cortex. The quantity of insoluable substances increases continually at the expense of the soluable ones. Jess‡ has stated that in age the insoluable albumenoids prevail in the lens at the expense of the soluble crystallins. He used the reaction of cystein, consisting in the fact that under the influence of a solution of nitroprussid natrium with a small quantity of ammoniak most albumins assume a red tinge. A similar red tinge can be brought about in the normal lens, while a lens opacified by age does not become colored, or only at the edge. The failure of the reaction is explained by the fact that the red tint is fixed by the crystallins and since these are absent in the center of the lens opacified by age, the coloring does not take place.

Chalupecky§ has repeated this experiment and has been able to confirm it. He has exposed the lenses of pigs' eyes for 3 hours to the influence of a quartz lamp. Changes took place similar to those in lenses opacified by age; the centre of the lenses remained under the influence of the nitroprussid without any tinting, a narrow line only became red.

I have repeated it by putting rabbits' and pigs' lenses in two vessels of 30 cm. under a quartz lamp. One of these was covered by a quartz plate, the other by one of euphos glass, which absorbs all ultraviolet rays of the quartz lamp. The thermometer did not rise above 26 degrees. After three hours the lenses under the quartz plate had still a slightly gray tint when compared with those under the euphos glass.

When the lenses are cut and then put into a nitroprussid solution, those under the euphos glass are colored totally, the others only at their edges. These experiments teach that the ultraviolet

*La Clinique Ophtalmologique, January, 1916.
†Zeithschr. f. phys. Chemie, XVIII, 1894.
‡Zeithschr. f. Biol., No. 61.
§Wien. med. Wochenschr., 1913, No. 31 and 32.

rays produce in the lens chemical changes analogous to those observed in lenses opacified by age. Chalupecky, further, found that in a three hour exposure to the light of a quartz lamp the less soluble albumins were increased by 13.3 per cent.

These facts render it necessary for us to again examine if the changes which take place in our lenses during life are not caused by the influence of the invisible rays.

It is most necessary to find out what influence these rays exert on the lens. I have at different occasions (the last time in *Graefe's Arch.* LXXXVI) shown with what an intensity the lens absorbs and changes the rays not directly visible. A large quantity of these rays is in the lens transformed into visible light, this is seen by the fluorescence of the lens; another portion is absorbed without the appearance of fluorescence. In that paper I have shown that a lens compressed to 3 mm. of a man 60 years old, absorbs all the ultraviolet rays.

The spectrum shows us the quantity of light absorbed by such a lens. The photochemic energy of the rays absorbed by a 3 mm. lens is exactly the same as that of all visible rays. The normal lens is thicker than 3 mm. in its centre and therefore absorbs more light than this comparison shows. In tissues rich in nerves and cells the chemical irritation of the light produces in the lighted area and its neighborhood reactions which paralyse the chemical irritation. The lens has no nerves and few cells and such counter-reactions are wanting. The effect of the light is purely chemical. Such a quantity of energy cannot be without effects. If we do not know them, we must hunt for them whereto these rays withdraw themselves from our observation.

Aside from absorption, there are still other circumstances which we must consider in the influence of light on the lens. One part of the light is reflected by the posterior surface of the lens. We can observe this light by oblique illumination. During every reflection the light loses above all the rays of short waves; the spectrum becomes shorter at its invisible end. This light is reflected by a concave mirror in the centre of the lens and against the anterior surface of lens which rejects it into the interior. It is rejected from one surface to the other as between two concave mirrors in the mass of the lens which with preference absorbs the short wave rays. This explains that light falling into the eye in the direction of the axis can influence parts of the lens which seem to be protected by the iris against this light.

We have to study another kind of effect of light in connection with the lens. What is the effect of diffusion of the light in the lens? Van der Hoeve has already studied this question. When a sun's ray penetrates into a dark chamber we see its entire course distinctly. Fine particles suspended in the air diffract the light. Even when the ray does not penetrate into our eye these diffracted rays make it visible to us. The diffraction of the light is much greater for the violet rays than for the red rays. The blue color of the sky is due to the stronger diffraction of the short wave rays. If our earth had no atmosphere or the atmosphere was "optically empty" the sky would appear black. Still the atmosphere appears blue. We can observe the same phænomenon if, in a dark room, we blow the smoke of a cigar into a ray of light. The smoke appears blue. The sky appears bluest at 90 degrees from the direction of the sun's rays. The principle of the ultramicroscope rests on this diffraction of the light. Horizontally to the axis of the ultramicroscope an intense ray of light is thrown through an object; if this is not "optically empty", the rays diffracted by the smallest particle are seen in the microscope. Particles so small that they defy every other observation can be seen. According to the laws of physics it is certain that the short wave rays participate most in this. This can be noticed in the microphotographs. Professor Kalkowsky has shown me such microphotographs in which things are shown much better than they seem to the eye. This is explained by the fact that the short wave rays still influence a photographic plate, when the eye cannot perceive them. The diffusion of light increases towards the short wave end of the spectrum. . . . . .

What is the influence of the diffusion of the light in the lens? Is the lens "optically empty?" In order to find out I have sent a thin intense ray through a lens in a dark chamber. In order to avoid the fluorescence the ultraviolet rays were cut off by an euphos glass. Yet, a very clear ray can be seen passing through the lens. The lens is, therefore, not "optically empty" but filled with numerous small particles which diffract the light. In this case again the short wave rays are most intensely diffracted. In fact this is what produces light on the parts of the lens which are covered by the iris. This light must be especially rich in short wave rays. A light of such a composition will act on the ciliary body like a blinding flash and cause pain in it, and, when this influence is prolonged, even inflammation.

We know now that short wave rays produce in the albumins

of the lens changes similar to those which we find normally in age. We have thus the explanation, also, how the short wave rays can influence the periphery of the lens. We have to ask ourselves now, are the hardening of the nucleus of the lens, the presbyopia due to it and cataract effects produced by the short wave rays? I am convinced that these changes represent the cumulative influence of these rays. Best* has stated that such a cumulative influence does not exist. He pointed to the retina which bears without damage a great deal of light and is only damaged by a very excessive illumination. This comparison is not correct. The retina is a tissue which is provided to a high degree with apparatus for the defence against intense light. With the lens which is composed of epithelial elements we think first of an analogy with the skin. In the skin we plainly see the cumulative influence of light which during life causes very visible changes. The skin is very rich in nerves and cells, short irritations by light are annihilated after a short time. In the lens without nerves and poor in cells such an annihilation does not take place. Here the chemical influence of light must be still more powerful.

*Deutsche med. Wochenschr., 1914, p. 144.

CONCERNING THE TREATMENT OF TABETIC OPTIC NERVE ATROPHY.*

(From Prof. Dr. L. Heine's Eye Clinic, Kiel.)

By Dr. C. Behr.

The enthusiasm aroused at first by Ehrlich's discovery of salvarsan which already looked forward to a cure of the metaluetic nervous affections as an accomplished fact, was soon followed by a reversion of feeling which went beyond the aim in the opposite direction and refused to acknowledge any therapeutic influence on these affections, thinking them incurable and irrepressible. Recently, however, a number of papers have shown that the previous hopeful views for these diseases too are not altogether without ground to go on, if the expectations for a result from the therapy are not raised beyond a sensible degree. A *restitutio ad integrum* when the disease is fully developed cannot be hoped for in view of the serious degenerative changes in the central nervous system. We will have to content ourselves with retarding the course and in the most favorable case to reach a lasting standstill or at least one which will last for some time. Such results seem to have been actually accomplished in some cases of tabes. The believers in the dogma of the incurability, however, assume that these were not true tabes cases, but cases of cerebrospinal lues with tabetic symptoms, pseudotabes syphilitica. Neglecting the fact that thus far an anatomical proof of such a statement is wanting a little thinking will show that such a differentiation cannot be brought to accord with our up-to-date bacteriological and clinical experiences.

The examinations of Noguchi, Graves, Uhlenhut, Mulzer and others are definitive and have not only proven the presence of living spirochaetæ in the cerebral cortex, the blood and in the cerebrospinal fluid of paralytics and tabetics, but, also, their full virulency through positive inoculations. From a bacteriological ætiological standpoint there is, therefore, no difference between cerebrospinal lues and metalues.

How can we, now, explain the difference in behavior to the therapy, in spite of the common ætiology: why, in general at least, will a cerebrospinal lues yield to treatment when until recently this seemed impossible with tabes? It seems to me that

*Klin. Mtsbl. f. Augenhlk., V. LVI, p. 1.

we are even to-day to a certain degree, if not altogether, able to answer this question. The cause is first the difference of localization of the spirochaetæ infection which in cerebrospinal lues is purely meningeal (if we neglect the lues of the vessels and circumscribed gumma formation), in tabes on the contrary it is chiefly in the true nervo-glious substance, and secondly in the different physiological method of the supplying of blood and the different metabolism in these two tissues.

The success or failure of a medicamentous therapy, in general, depends above everything else on how much of the specifically active remedy when introduced into the body can be brought into the disease foci. Since this introduction takes place by way of the blood it will depend especially on the blood supply of the diseased organs, the better the blood supply the more favorable are the conditions for the effectiveness. There are, however, great differences in this direction between the mesodermal meninges and the ectodermal brain substance, as is best seen in the optic nerve. This nerve as a part of the brain is surrounded by the comparatively wide pial sheath carrying the largest part of the nutrient vessels and from which direct offshoots in the form of septa traverse the whole of the nerve. With these and enclosed within them the smaller bloodvessels, also, reach the interior of the nerve for the nutrition of the nervo-glious substance proper. This is, however, separated all around from the connective tissue by glious membranes (membranæ limitantes gliæ). Nutrition by a direct route, in which the nutrient substances pass directly from the bloodvessels into the tissue, is, therefore, out of question. The uniform distribution of the nutrient substances within the single nerve fibre bundles takes place through certain glia fibres which on one side are connected with the limiting membrane and on the other side are in direct anatomical contact with the axis cylinders and the marrow sheaths. The nutrient substances coming out of the bloodvessels must first pass through the limiting membranes and wander through the system of glia fibres for a longer or shorter distance before reaching the nervous organ to be nourished—the axis cylinder or the ganglion cell—while the meninges are nurished immediately by the bloodvessels. Although this great difference between the meninges (and their continuation) and the nerve substance proper is adapted to the physiological necessities of the two tissue forms, under pathological conditions the relative inferiority of the blood supply and of the parenchymatous

lymph current must make itself felt in the brain substance in that there is less resistance to the immigration of pathogenic bacteria.

The nutritive conditions within one and the same nerve fibre bundle vary according to the position of the fibres. Since the nutritive fluid enters from the periphery of the bundle, in the septa, concentrically from all sides towards the axis, the fibres lying most axially are, of course, in the most unfavorable condition because the concentration of nutrient material in the lymph must naturally get less toward the axis of the several bundles. This defect can be remedied by an increase in the concentration of lymph coming from the bloodvessels and by increasing the current of the tissue fluids. This defect must show itself, however, in a different manner when therapeutic agents are taken into the body. Especially with the specifically acting remedies the therapeutic dosis is very close to the toxic dosis and thus the possibility of reaching the effect by an increased concentration is materially restricted. Let us assume that a nerve fibre bundle is uniformly permeated by spirochaetæ in its whole transverse section, then the specifically acting remedy when introduced into the body (that is one which by a chemical attachment kills the microbes), will at once be bound to the spirochaetæ when it leaves the septa. The nearer the axis the less concentrated it must be in the tissue fluid. Below a certain concentration, however, the remedy cannot produce a killing effect, on the contrary its action is directly reversed, as we know, for instance of salvarsan. Thus the killing remedy is changed into a stimulating one, which instigates the spirochaetæ to an energetic proliferation (provocating injection of salvarsan). With a uniform distribution of the germs in the transverse section of a nerve fibre bundle we can readily understand that under these anatomico-physiological conditions, aside from their death, a proliferation of the spirochaetæ takes place, which cannot only annul the healing effect of the remedy but in unfavorable conditions, for instance, when the number and the virulence of the micro-organisms are very great, may make conditions decidedly worse. On the other hand, in the meninges such a mobilizing effect of salvarsan is not to be expected—assumed that the total dosis given was sufficient—since here the foci are in direct contact with the vascular system.

Aside from the introduction of the remedial agent, the quickly produced innocuousness and the removal of the toxic material coming from an infection focus, play the largest rôle in fighting

an infection. This takes place through the creation of antibodies which chemically neutralize the toxins and then through the quickest possible removal into indifferent and resistent organs by means of the lymph channels. The former depends, also, on the blood supply. The large defects which we had to point out as working against the keeping up of a uniform sufficient concentration of a remedial agent for the nervo-glial tissue as contrasted with the meningeal act here in the same way. But, also, concerning the quick elimination of the toxic substances there are differences between the two kinds of tissue which take on a special importance: In the meninges the tissue fluid enters immediately into the perivascular spaces of the veins or into the subdural, or rather subarachnoidal spaces. No special serious damage is here to be expected from the toxins alone, particularly since the connective tissue possesses a greater power of resistance against them. In the nerve fibre bundles of the optic nerve the anatomical and physiological conditions of which we can on account of its developmental position at once apply to the remainder of the central nervous system, the elimination of the used-up nutrient material does not, or at least not in the main, take place through the perivascular lymph spaces of the septal vessels. On the contrary the tissue current flows within the single nerve fibre bundles centralward into the cranial cavity and comes out here only through the pial sheath into the subarachnoid and subdural lymph spaces. Toxins formed in the nerve by a spirochaetæ focus will be excreted at this place only. The more peripherically this focus is situated, the longer the way by which the poison can get into immediate touch with the nerve fibres. It is at least probable that in this manner degenerations and disturbances of function may be produced where there are no spirochaetæ. In my opinion this, also, explains in part the findings so extremely frequent in tabetic optic nerve atrophy of an irregular concentric contraction of the visual field which points to degeneration of the peripheral nerve fibre bundles. As I have been able to demonstrate, there is just at these places a main collecting basin and an organ for the removal of the free tissue lymph (subpial lymph space system).

From what we have said lues cerebrospinalis is due to a spirochaetæ infection of the meninges and their appendages, tabes dorsalis and paralysis are due to an infection of the cerebral substance itself. The partial lack of influence of our therapy on the two last affections is explained by the insufficient blood supply

which renders impossible a uniformly strong and sufficient concentration of the remedy to kill the spirochaetæ and by the especial behavior of the parenchymatous lymph stream which does not permit of a rapid destruction and excretion of the toxins formed by the spirochaetæ.

From these view points a material difference between lues cerebrospinalis and metalues can no longer be maintained. In proof we have those cases in which both processes exist side by side. A case of Pick's (Festschr. f. Ph. I. Pick, 1898, Wien and Leipzig) with anatomical examination is especially instructive in this direction. During life reflex immobility of the pupil, Westphal's phænomenon, Romberg, ataxia, disturbances of sensibility, neuritis optica. On dissection: Meningitis syphilitica chronica cerebri and cerebral sclerosis in the sacral spinal chord.

I think the time in which the therapy acts in these two groups is, also, of importance. In accord with the mostly rapid development of cerebrospinal lues the subjective disturbances are more severe and the several symptoms develop rather rapidly to their complete height. The patient consults a physician very rapidly and the therapy is early begun before more extensive and more severe anatomical changes have been formed, before the spirochaetæ have taken a firm hold on the tissues. In the so-called metalues the conditions are exactly reversed. Aside from the fulminant cases, the development is usually slow and insinuating. Before objective symptoms or subjective disturbances are noticed, extensive degenerations are already formed in the brain and spinal chord. The same occurs in a typical manner in the optic nerve. It may actually be considered the rule that first of all the symptoms of an atrophy of the optic papilla appear while the functions (central vision, visual acuity and color sense) are still perfectly normal (Uhthoff, Wilbrand and Saenger). Their disturbances develop later only. The reverse condition—that functional disturbances are present without atrophy or precede it, according to Uhthoff—speaks directly against a tabetic base for these changes.

Everything points to the fact that if a tabetic seeks the physician his affection, purely anatomically considered, has progressed much farther than the objective findings and the subjective disturbances permit us to perceive. The spirochaetæ have had time to settle themselves undisturbed just in those localities in the nervous parenchyma of the brain and spinal chord, which are incompletely only accessible to the natural defensive forces of the

organism and thus, also, to the ingested artificial remedial agents.

These considerations, too, are likely to explain the actual contrast of the two disease groups towards the therapy as being only apparent. However, we do not want to deny that other factors may play a rôle with which we are not yet acquainted. I think here of the cases in which several men infected at the same source are affected by paralysis only, in others by tabes only; furthermore of the not exactly rare cases of conjugal tabes, or paralysis. Such observations seem to show that the special strain of spirochaetæ is of importance for the later development of metalues.

Through these considerations the difficulties encountered by the therapy of tabes, or paralysis are easily explained. The farther the anatomical process has gone the more unfavorable is the prognosis. Only the very first stages can give hope of success with the modern syphilis therapy. The whole question of the curability of tabes or paralysis depends on as early a diagnosis as possible.

In my article "On the behaviour and the diagnostic value of the dark adaptation in the different diseases of the optic nerve," published in this Journal, I have pointed out that we cannot do full justice to this postulate by a general neurologic method of diagnosis. With the ophthalmological method this is different. I could draw attention to the fact that by means of the dark adaptation we can prove a beginning tabetic degeneration of the optic nerve at a time when the ophthalmoscope cannot detect any disease. This disturbance of function which may be looked upon as the most regular symptom accompanying tabetic optic nerve atrophy is explained as the direct consequence of the primary syphilitic focus in the nerve. According to Wilbrand and Saenger's anatomical researches the optic nerve atrophy in tabes does in the majority of the cases not occur diffusely, but very circumscribed in the periphery of the nerve. Here at the very first beginning a focus-like degeneration of fibres is seen which disappears completely centrally and peripherally from this point and which is explained as the direct consequence of the action of the toxins of the spirochaetæ lodging here. In this place the interruption or retardation of conduction of the fibres which regulate the dark adaptation occurs. The ophthalmoscopically visible atrophy of the nerve is only indirectly connected with this functional disturbance, it is only the sign that the degeneration of the fibres has wandered from the primary focus into the pa-

pilla. This explains why the disturbance of dark adaptation must be in existence for a prolonged time before the evolution of an ophthalmoscopically visible optic nerve atrophy. By this method we have become possessed of a means to discover the tabetic degenerative process in the optic nerve in its very first stages. No other method, not even one of general neurology, is so fine and certain.

To this we may add that the eye symptoms, especially reflex immobility of the pupil in its classic form (miosis, lack of light reaction, increased convergence contraction), may exist in many cases and for a long time as the only signs of tabes to which the others after years or decades only are added, usually in quicker succession. These monosymptomatic or oligosymptomatic cases which are stationary for a long time teach us that tabes per se is not of an absolutely progressive character. They are explained by the fact that the natural forces of defense in the body can repress the infection for a prolonged period. On these cases we may base the hope that we may be able to favorably influence tabes therapeutically and even bring it to a standstill.

As this diminution of dark adaptation is the finest diagnostic agent in tabetic optic nerve disease it serves us to learn in figures the degree of effect of our therapeutic measures against this process. Numerous observations have shown us that the sensibility to light often recovers to a considerable degree under antisyphilitic treatment, while the existing ophthalmoscopic changes or the disturbances of the central visual acuity, of the color sense or the field of vision, do not show the slightest improvement. From this we may probably conclude that, at least, at the beginning of the affection the damage to the fibre apparatus governing the dark adaptation is not due to an incurable, absolute interruption, but to a toxic retardation.

A similar behavior I have, too, often observed in acute inflammatory processes in the optic nerve. In these cases there exists, aside from neuritic symptoms at the papilla, simply a diminution of dark adaptation to a higher or lesser degree while visual acuity and visual field are normal. After the disappearance of the inflammation and the formation of the neuritic atrophy of the papilla the dark adaptation again became normal. These cases, also, show that the atrophy of the optic nerve by itself is not the cause of the disturbance in adaptation. The following case may serve as a paradigma.

F. H., 55 years old, paper hanger. Denies infection. Lan-

cinating pains. Incontinence of urine. April 10th, R. V.=6/20 to 6/15; L. V.=6/7. R. papilla gray white, a trace of red. L. temporally gray white, nasally red. Field: R. sector defect nasally and down; L. slight concentric contractions for white, for color relatively normal. Pupils normal. Patellar and Achilles tendon reflexes wanting. Disturbances of sensibility in the area of the peronei. Wassermann reaction (blood) positive. Dark adaptation after three-fourths hours: R. 64 E. E.; L. 744 E. E. (E.E.=Empfindlichkeitseinheiten, units of sensibility). Inunctions at home. May, 1910. No change, except D.A. after three-fourths hours R.=659 E.E.; L. 2067 EE. No. treatment. In September, D.A. down in R. to 81 E.E.; L. 971 E.E. Salvarsan 0.6. October, D.A. R.=659; L. 1784 E.E. No treatment. December, D.A. R.=106; L. 265 E.E. January, calomel cure (40 per cent.), then D.A. R. 659 and L. 934 E.E.

Later the improvement in dark adaptation grew continually less, till finally the therapy had no more effect. The tabetic process went on uninterruptedly to complete amaurosis in 3 years. This case shows how by an antisyphilitic treatment regularly a very considerable improvement was produced in the dark adaptation while the disturbances in the other visual functions remained unaltered. Although the improvement lasted always a short time only it shows that the tabetic process as such is amenable to therapeutic influence. The momentary and not very great improvement further shows that our remedial agents and especially the manner and timely extent of their effect are at present still unsatisfactory. Further study must find the right ways. It is clear that these cannot alone be experimental pharmacological aims. Cases, like the one reported, teach us that with the present remedies successes may be reached. It will be more important to clear up the physiological relations, especially the kind and course of the parenchymatous lymph stream, and to find ways and means to stimulate and to increase it in order to enhance the concentration of the remedial agent.

I want to say here that in general the effect of the different agents as judged by the improvement in dark adaptation are of equal value (calomel, mercury, salvarsan).

How do we explain it that the dark adaptation can be considerably improved by the specific therapy while the other visual functions (acuity, field, color sense) remain uninfluenced?

Concerning this I have to revert to my former paper in which I detailed the anatomical and physiological fundaments of dark

adaptation as far as the optic nerve is concerned. The function of dark adaptation, that is the regeneration of the visual substances on which it is based, must not be looked upon as process limited to the retina; it takes places rather in the form of a glandular process, that is, it is regulated by reflex. The reflex arc lies at the basal visual tract, its centre probably in the lateral ganglion geniculatum. It is probable that not every single element of the retina which secretes these visual substances is firmly attached to one single centrifugal secretory fibre, but always to a larger number, a larger retinal area. This alone explains that this system is so easily damaged. To this must be added that the dark adaptation is much more easily affected by toxic and chronic degenerative conditions in the nerve, than the other visual functions. The focus which produces these toxic substances need not be large. On account of the peculiar arrangement of the parenchymatous lymph stream these substances are divided pretty uniformly over the whole transverse section and are able to rescind the conductibility of the fibres at greater distances from the focus.

On the other hand every single cone as an organ of daylight vision communicates by a special fibre with a perceiving element in the optical centre. The destruction of a single cone or of its centripetal tract does not reach consciousness. The loss can be proven only when a larger number of fibres lying immediately side by side are destroyed. In the beginning at least the tabetic process shows throughout an elective character. We find in one fibre bundle single degenerated fibres by the side of perfectly normal ones. If, therefore, we notice a disturbance in the function of the cones we can immediately conclude from it that there is an extensive degenerative process going on in the optic nerve.

This explains, too, that disturbances of the visual acuity, color sense and visual field in tabes are in general hardly influenced by therapy.

In the paper mentioned I reported some cases of neurologically certain tabes dorsalis in which as far as the eye was concerned nothing was noticed, but a disturbance of the dark adaptation. All other functions and the ophthalmoscopic appearances were normal. Then, before our eyes, in a shorter or longer period, a plain atrophy of the disc developed with consequent disturbance of the function of the cones. The disturbance of the dark adaptation is therefore the first symptom of a tabetic degeneration of the optic nerve.

Naturally such cases are very rare and can be found only when in connection with other clinics, all tabetic cases are systematically and also ophthalmoscopically examined. I can report one such case in which by energetic and continued antiluetic treatment the opticus process was brought to a standstill.

M. H., 39 years old, laborer. Infection 1897. For years lancinating pains in the legs, belt sensation, paræsthesiæ in hands. V.=6/5 both. Pupil narrow, not quite round. Light reflex sluggish; convergence reaction normal. Movements free. Papillæ slightly pale, more in R. Dark adaptation: After three-fourths of an hour in the dark R.=331, L.=625 E.E. General examination in medical clinic. Diagnosis: Tabes dorsalis; lack of patellar and Achilles' tendon reflexes. Disturbances of sensibility. Romberg positive. Wassermann in blood negative. Calomel cure. Two weeks later D.A. R.=644 and L.=1309 E.E. Ambulant inunction cure. Five months later D.A. R.=1001, L.=2183 E.E. The right papilla is now pale so that the pathological base of the discoloration can no longer be doubted. Seven months later D.A. R.=2571, L.=4329 E.E. The other symptoms have remained unaltered. The light reaction of the pupil seemed improved. The paleness of the papilla had not progressed further, field and acuity remained normal.

The patient remained under my observation for 3 further years. The dark adaptation remained equally high. The other symptoms were unchanged.

While at the beginning of this observation a high grade disturbance of the dark adaptation proved that in this case, which from the general neurological findings must be considered as tabes, the optic conducting apparatus must also have been diseased; this was made absolutely certain by the increasing paleness of the right papilla. In the paper mentioned above I have pointed out that the course of the optic nerve affection can be divided into four phases. In the first, there is a high grade disturbance of the dark adaptation; the sensibility of the retina increases but slowly during the stay in the dark, and the maximum of the sensibility which the eye can reach remains considerably under the low normal limit, which may be accepted as 1500 E.E.

At this stage the ophthalmoscopical findings are absolutely normal, the central acuity and the field are undisturbed.

In the second phase a plain atrophy of the papilla of the optic nerve is added to the disturbed dark adaptation. In the third

phase the visual acuity and the visual field begin to retrograde, progressing to the fourth phase in which there is total amaurosis with atrophy of the optic nerve.

In our case the treatment was begun in the first phase. The degeneration descending from the different tabetic foci causing the disturbance in dark adaptation in the optic nerve had hardly reached the papilla, which, while it was pale, could not be considered as pathologic. Through the treatment the dark adaptation was markedly improved and remained at a normal height for 3 years. I do not think there can be any other explanation but that the pathological process was brought to a standstill, also, at the location of the primary focus by complete destruction of the spirochaetæ. Since, however, at the locality of this focus numerous fibres had been destroyed, we cannot wonder that by descending degeneration of these interrupted fibres an atrophic discoloration showed itself at the papilla, so that in spite of the improvement in function an increase in the objective symptoms resulted. This is, however, not unusual. Thus we have in acute retrobulbar neuritis, for instance, in its beginning no ophthalmoscopic changes in spite of serious changes in the visual field and central visual acuity. After the healing then, in spite of improvement in function, through descending degeneration of the fibres destroyed in the locality of the focus, atrophy of the papilla occurs.

I believe, therefore, to be right in assuming that in the case reported a beginning tabetic process of the optical conducting tract has been brought to a stillstand, or rather a cure, through specific therapy, for its existence was proven by the later paleness of the papilla; on the other hand we may speak of cure, since the improvement remained constant during observation lasting 3 years.

These observations show that even to-day the tabetic process can be influenced by therapy. For, even if the only functional disturbance caused by tabes can be cured, the possibility of an improvement or cure of the whole tabetic process, and with this probably of metalues, seems proven. Therein lies the practical value of our observation.

(*Continued in September Number.*)

INFORMATION REGARDING EXAMINATION IN OPHTHALMOLOGY.

The American Board for Examinations in Ophthalmology has been established to examine and certify the preparation of those who intend to practice Ophthalmology chiefly or exclusively. Its members are chosen by the Section on Ophthalmology of the American Medical Association, the American Ophthalmological Society and the American Academy of Ophthalmology and Oto-Laryngology; and after 1920 its certificate will be required of all applicants for membership in the two latter organizations.

WHO WILL BE EXAMINED.

Graduates of at least two years standing from reputable medical colleges who present evidence of the study and practice of Ophthalmology sufficient to give them a working knowledge of the subject, and who present evidence of satisfactory ethical and medical standing in their professional community will be examined. Those who have ten years of practice in Ophthalmology must submit evidence of a legal medical degree, all medical papers they have published, and evidence of satisfactory ethical and medical standing in their professional community. Those who have practiced Ophthalmology from five to ten years must in addition submit evidence of service in an Ophthalmic hospital or clinic, or with an Ophthalmologist in private work, or in lieu thereof reports of cases and statement of operations performed. Those who have practiced Ophthalmology less than five years must give evidence of one year of hospital or laboratory work following four years work in a graduate medical school, a year of service in an Ophthalmic hospital, clinic, or assistantship in private Ophthalmic practice.

APPLICATIONS FOR EXAMINATION.

These data must be submitted on the special blank prepared for the purpose *at least three months before the date of examination to be taken,* and must be accompanied by evidences of all education received and work done, in preparation for a pursuit of Ophthalmic practice, together with a signed photograph of the applicant, and bank draft or money order for the examination fee. The evidences of clinical work must include the diagnosis of fifty cases that have been under the applicant's observation and care.

FEES.

The examination fee is twenty-five dollars. The draft or money order for it should be drawn to the order of "The American Board of Ophthalmic Examinations." If the applicant is not admitted to examination the fee will be returned. If he takes the examination and fails, he will be admitted to a second examination within three years, without additional fee, but must give sixty days notice of his intention to appear for re-examination.

EXAMINATIONS.

The applicant must send to the Secretary of the Board *at least sixty days before the date of examination* reports of five cases that he has attended. These must be important cases of ocular disease or defect, and of varied character. The reports of them should be as complete as possible with regard to all important points. They will be studied by the examiners, who may use them as a basis for oral examination. At the date for examination, and so many succeeding days as may be necessary, the candidate must present himself at the place of examination prepared to undergo clinical, oral, written and laboratory examinations.

CERTIFICATES.

As soon as practicable after the close of an examination, candidates will be notified of the result, and to those who have successfully passed a certificate signed by the members of the Board will subsequently be issued.

F. C. Todd,
Secretary of the Board,
Minneapolis, Minn.

ABSTRACTS FROM MEDICAL LITERATURE.

By W. F. Hardy, M.D.,
ST. LOUIS, MO.

THE THEAPEUTIC ACTION OF IODINE.

There are many subjects and problems not purely ophthalmological which are yet of intense interest and more or less practically associated or allied to ophthalmology. We are prone to accept well established customs and statements without a deeper inquiry into the why and wherefore of them. It is common

knowledge that iodides cause resorption of exudates and gummatous material or at least are given for that purpose. Jobling and Peterson (*Archiv. of Internal Medicine*, Vol. XV, p. 286) sought to find an acceptable explanation. Time was and not so long since when we spoke of iodides and other drugs as alteratives, whatever that may have meant. Results obtained with the iodides have been ascribed to the influence on general metabolism; their supposed action in causing a fall in blood pressure; to a change in viscosity of the blood; to lymphocytosis; to an increased activity of the lymphatics and to the oxidizing properties of the nascent iodine. The absorption, distribution and excretion of iodine has been widely studied. The authors confined their investigations of the action of iodine to its influence on the antitrypsin of the blood tissues. They believe that the antitrypsin is the most important factor in preventing the resolution of necrotic tissues such as are found in infarcts and in the caseous areas of syphilis and tuberculosis. The ferment inhibiting action of serum is due to the presence of compounds of the unsaturated fatty acids which constitute the antitryptic element of the blood. This suggested to the authors that the action of iodine in the body may be due to a combination with the fatty acids, thus causing a neutralization of the activity of these substances as ferment inhibiting agents. This lowering of the antienzyme strength of the blood tissues, permits of the removal of dead tissues by autolysis. A series of careful experiments showed that iodides given to human beings caused a very considerable reduction in the antitryptic activity of the blood. In two cases in which iodism was produced the antiferment was much higher than it had been during the course of treatment. It was shown that serum deprived of its antitrypsin becomes toxic for the species from which it was obtained. Clinicians have the impression that iodides are harmful in tuberculosis due to the consequent softening of the tubercles. Several observers have refuted this. Loeb and Michaud observed that a tuberculous rabbit's eye contained twice as much iodine as was present in the normal one. Other authors conclude that the large amount of iodine present in necrotic tissues, whether tuberculous or otherwise, is dependent on purely physical conditions and not on a chemical attraction or physical absorption.

Iodides have no effect on the infecting organism of syphilis, their administration not affecting the development of experimental lues in animals. Iodine does penetrate into syphilitic

tissues. Iodides are effective in tertiary lues when necrosis of tissues is much in evidence and are of little value in the early stages of syphilis. According to the authors' view "iodine neutralizes the action of the agents which prevent solution and absorption of necrotic tissue, and at the same time lays bare to the action of the real germicidal agent the infecting organism which previously had been protected by the necrotic tissue. With the exposure of the infecting organism such agents as mercury and salvarsan would be much more effective."

STATISTICAL NOTES ON GLIOMA RETINÆ, WITH A REPORT OF FORTY-ONE CASES.

This paper by Berrisford, of St. Paul, Minn., is contained in the *Royal London Ophthalmic Hospital Reports,* Vol. XX, Part II, March, 1916. The object of the study was to complete the collection of cases and reports inaugurated by Collins and Lawford and carried on by Marshall and later Owen. Some of Berrisford's material was gathered from private sources, the greater part coming from the Royal London Ophthalmic Hospital. The various phases of the subject are separately considered. The gist of the notes may, however, be gleaned from the summary. (1) Relative frequency.—Taking the total number of new patients at the Royal London Ophthalmic Hospital during the last forty-two years the ratio of glioma retinæ to other eye diseases is found to be 1 to 9,614, or slightly more than 0.01 per cent. (2) Sex.—In the series of 41 cases on which this paper is based there were 22 males and 17 females; in two cases the sex was not recorded. In reviewing the literature on this topic, statistics show that glioma retinæ occurs more often in males than in females, but the difference is too small to be worth considering. (3) Eye affected.—The right eye was affected alone in 16 cases, the left eye in 17 cases. There were six bilateral and 1 doubtful bilateral case. In one instance the eye involved was not recorded. The proportion of bilateral to unilateral cases is in this series 1 to 7, or 14.6 per cent. The usual figure quoted is 25 per cent., in accordance with Wintersteiner's report of 405 cases. (4) The age of the patient when the growth was first observed.—The tumor was in three cases first observed at birth, in 4 cases during the first three months of life, in three cases between the third and sixth months, in two cases between the sixth and

twelfth months, in six cases during the second year of life, in three cases during the third year of life, in four cases during the fourth year of life, in three cases during the fifth year of life, and in two cases during the sixth year of life. Of 135 selected cases 41 per cent. showed evidence of the presence of glioma retinæ during the first year of life. *Recoveries.*—In this series 9 patients may be considered as cured, three years having elapsed since the enucleation of the gliomatous eyes without signs of recurrence. *Fatal cases.*—There were four fatal cases, in two of which there was a considerable involvement of the optic nerve and an extension of the growth into the orbital tissues. In the other two cases the growth was bilateral, and in both instances the parents would not consent to double enucleation. It is noteworthy that of the 9 cases which have recovered, in not one had the growth invaded the optic nerve as far as its cut end. This shows the importance of cutting the nerve as far back in the orbit as possible. *Gliomata in Shrunken Eyes.*—The presence of glioma in a shrunken eye is a rare condition, only 20 cases having been hitherto recorded in ophthalmological literature. The present series contains one case of this type. *Family History.*—While it is comparatively rare to find more than one member of a family affected by glioma retinæ, there are only two instances to be found in literature where a child once affected with glioma retinæ has grown up and has had children who developed the same disease. In the present paper the appearance of this disease in another child belonging to one of the above families (the Grover family), is reported for the first time, and the pedigree of this family is given in full. No fresh cases of this kind have, however, been met with.

BOOK REVIEWS.

26TH ANNUAL REPORT OF THE EYE, EAR, NOSE AND THROAT HOSPITAL OF NEW ORLEANS, LA.

In this excellent report which shows the large volume of work done at this well known Hospital I find the following remarks which refer to the practice of Drs. H. Dickson Bruns and E. A. Robin:

"For the past nine years, we have been treating a large pro-

portion of our patients, operated on for cataract and other serious eye affections, as ambulants; i.e., immediately after the operation, they are allowed to go to their homes, with instructions to report to the hospital on the following day and every day thereafter until discharged. This method presents several advantages. We have found it not only quite practicable, but decidedly in the best interests of the patients.

"The notable absence of post-operative insanity among our cataract patients, since its adoption, leads us to believe that the very fact of confining them in hospital beds, amid unusual surroundings, is an important factor in the ætiology of this very serious and sometimes fatal complication."

Since I have practiced the same method for many years, when circumstances seemed to demand it, and at one time for a period of ten years in all cases, I feel that I am compelled to add my testimony to theirs. From my experience I know that in many cases the patients are better off, keep in good spirits and come out with the same results as those which are operated on in hospitals. The fear of injury from moving is to say the least exaggerated. This fear so dominates the profession that their attitude forced me to give it up as a rule of practice. Yet I know that when necessity and the patient's conditions seem to demand it, it is just as good and sometimes a little better, than to force them to stay at a hospital, especially in a ward.

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